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Accuracy of the pacemaker-mediated tachycardia algorithm in Boston Scientific devices

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Abstract

Introduction: The incidence of pacemaker-mediated tachycardia (PMT) varies as a function of patient characteristics, device programming and algorithm specificities. We investigated the efficacy of the Boston Scientific algorithm by reviewing PMT episodes in a large device population.

Methods: In this multicenter study, we included 328 patients implanted with a Boston Scientific device: 157 non-dependent patients with RYTHMIQ™ activated (RYTHMIQ group), 76 patients with permanent AV-conduction disorder (AV-block group) and 95 Cardiac Resynchronization Therapy patients (CRT group). For each patient, we reviewed the last 10 remote monitoring-transmitted EGMs diagnosed as PMT.

Results: We analyzed 784 PMT episodes across 118 patients. In the RYTHMIQ group, the diagnosis of PMT was correct in most episodes (80%) of which 69% was directly related to the prolongation of the AV-delay associated with the RYTHMIQ algorithm. The usual triggers for PMT were also observed (PVC 16%, PAC 9%). The remainder of the episodes (20%) in RYTHMIQ patients and most episodes of AV-block (66%) and CRT patients (74%) were incorrectly diagnosed as PMT during sinus tachycardia at the maximal tracking rate. The inappropriate intervention of the algorithm during exercise causes non-conducted P-waves, loss of CRT (sustained in six patients) and may have been pro-arrhythmogenic in one patient (induction of ventricular tachycardia).

Conclusion: Algorithms to minimize ventricular pacing can occasionally have unintended consequences such as PMT. The PMT algorithm in Boston Scientific devices is associated with a high rate of incorrect PMT diagnosis during exercise resulting in inappropriate therapy with non-conducted P-waves, loss of CRT and limited risk of pro-arrhythmic events.

Keywords: Clinical: implantable devices – pacemaker-bradyarrhythmias; Clinical: implantable devices – biventricular pacing/defibrillation; Clinical: implantable devices – ventricular tachycardia/fibrillation; Clinical: implantable devices – physiologic pacing; Clinical: cardiac mapping – electrogram analysis

Introduction

Pacemaker mediated tachycardia (PMT) describes a repetitive sequence of sensed retrograde atrial events followed by ventricular pacing. This phenomenon has been observed and understood early after the introduction of dual-chamber pacing in patients presenting with intrinsic ventriculo-atrial (VA) conduction [1–3]. The majority of patients are asymptomatic but patients may report symptoms

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ranging from palpitations, rapid heart rates, lightheadedness, syncope, or chest discomfort. Prolonged PMT may be poorly tolerated or even lead to cardiac decompensation in patients suffering from underlying heart disease [4].

The incidence of PMT varies as a function of patient characteristics, programming of the device and the specificities of the algorithms provided by the manufacturers [4]. While many isolated clinical cases of PMT have been described, there is few data on the incidence of PMT across pacemaker and defibrillator recipients [5–9]. Depending on the manufacturer, one must understand the specific means of prevention, diagnosis and termination of PMT in order to optimize the management of device recipients. Each device manufacturer has a proprietary algorithm to detect and terminate PMT, with a large proportion of similarities, including the phases of suspicion, confirmation and termination. Studies to compare the respective effectiveness of these various algorithms of detection and termination of PMT are non-existent. In Boston Scientific devices, episodes of PMT are recorded in the memory. The ability to view the electrograms enables identifying and resolving the trigger of the tachycardia but also offers the opportunity to assess the frequency of this problem. Recently, we observed that their AV Search+ algorithm, which increases the atrioventricular delay in search for intrinsic conduction, was associated with PMT induction in a selected group of patients [10]. In the present study, we assessed the diagnostic and therapeutic efficacy of the PMT algorithm by reviewing EGMs of PMT episodes in a large population of implanted patients.

Methods

In this multicenter study, we included 337 patients implanted with a Boston Scientific device across four French centers. We distinguished 3 groups: 157 patients with a dual chamber device, without permanent AV conduction disorder and with the RYTHMIQ™ algorithm activated (RYTHMIQ group), 76 patients with a dual chamber device, permanent AV conduction disorder and RYTHMIQ™ algorithm not-activated (AV-block group) and 95 patients with a triple-chamber Cardiac Resynchronization Therapy device (CRT group). To be enrolled, patients needed to be programmed in AAI with VVI Backup (RYTHMIQ group) or DDD(R) mode (AV-block and CRT groups). When available, we reviewed the last 10 remote monitoring-transmitted EGMs labeled PMT with focus on determining the specificity of the algorithm and the etiology of the events. All patients gave written, informed consent for analysis of the data providing from remote monitoring and patient information was de-identified prior to analysis of the episodes.

Description of the anti-PMT algorithm

Boston Scientific devices label and save an episode as PMT when two criteria are met.

1. Sixteen consecutive ventricular pace (VP) and atrial sense (AS) cycles occur at the programmed maximum tracking rate. Default value of 130 beats per minute (coupling interval of 430 ms).
2. The variance between ventriculo-atrial intervals does not exceed 32 ms.

There exists no confirmation phase in Boston Scientific devices. Directly after diagnosis of PMT, the algorithm executes an automatic lengthening of the PVARP to 500 ms following the 16th cycle for a single cycle.

RYTHMIQ algorithm with AV search+

The RYTHMIQ™ algorithm, implemented in Boston Scientific dual-chamber pacemakers and implantable cardioverter-defibrillators, is designed to minimize ventricular pacing by favoring intrinsic ventricular conduction in non-pacemaker dependent patients. When RYTHMIQ is activated, the device operates in AAI(R) mode with VVI back-up. RYTHMIQ executes a mode switch to DDD(R) mode when AV-conduction disorder is suspected. In DDD(R) mode, the algorithm uses the AV Search+™ to periodically check for return of intrinsic conduction. After a certain number of cardiac cycles in DDD mode (default value at 32 cycles), the AV Search Interval is activated and the AV delay is extended to the programmed AV Search+™ value (default value: 300 ms). If a V-sense is detected within the 8 cycles period of AV Search+™, hysteresis continues. The AV Conduction Detector Counter is initialized at "0" and is incremented by each ventricular sensed event (marked as VS-Hy). When the AV Conduction Counter has reached 25, sustained conduction is detected and the device switches back to the primary AAI(R) mode with VVI back-up. The device maintains DDD(R) mode and does not switch back to the AAI(R) mode when no intrinsic conduction is detected within the first 8 cycles search period or when two out of the last 10 ventricular events are paced (sliding window).

Statistics

Continuous variables were expressed as median [min–max] and categorical variables were expressed as absolute numbers (percentages). Differences in baseline characteristics between groups were evaluated with the use of the Kruskal–Wallis H test for continuous data and Pearson's chi-square test for categorical data. Statistical significance was assumed at $p < 0.05$. Statistical analyses were performed using SPSS version 18.0 (SPSS Inc., Chicago, IL).

Results

Demographics and device programming

Patient demographics and device specifications are specified per group in Table 1. All CRT patients were equipped with ICDs while 17% of AV-block patients and 25% of RYTHMIQ patients were pacemaker recipients ($P < 0.001$ for comparison between three groups). CRT and AV block patients had a median cumulative ventricular pacing percentage of 99%, while RYTHMIQ patients had only a median of 1% cumulative ventricular pacing ($P < 0.001$ for comparison between three groups). The

Table 1
Demographics and device specifications of patients with dual chamber Boston Scientific devices divided in three groups according to underlying etiology. Categorical variables are expressed as absolute numbers (percentages), continuous variables are expressed as median [min–max].

	RYTHMIQ	AV-block	CRT	*P*-value
Patient demographics	*n* = 157	*n* = 76	*n* = 95	
Age [years]	68 [23–95]	75 [1–96]	68 [44–89]	<0.001
Male sex [*n*]	101 (64%)	49 (65%)	70 (74%)	0.319
Device type				
Pacemaker [*n*]	40 (25%)	63 (17%)	0 (0%)	<0.001
ICD [*n*]	117 (75%)	13 (83%)	105 (100%)	
Device programming				
Lower rate limit [bpm]	55 [35–80]	60 [40–80]	60 [40–70]	<0.001
Maximal tracking rate [bpm]	130 [100–155]	130 [110–170]	130 [110–160]	0.172
Minimum sensed AV delay [ms]	65 [60–220]	65 [50–220]	100 [55–200]	<0.001
Maximum sensed AV delay [ms]	150 [100–350]	150 [80–340]	120 [70–200]	<0.001
Minimum paced AV delay [ms]	80 [80–220]	80 [50–220]	140 [80–200]	<0.001
Maximum paced AV delay [ms]	180 [150–350]	180 [80–340]	160 [100–300]	<0.001
Device output				
Percentage ventricular pacing [%]	1 [0–100]	99 [0–100]	99 [0–100]	<0.001
Percentage atrial pacing [%]	18 [0–100]	25 [0–100]	8 [0–100]	0.023
Median respiratory rate [per min]	16 [11–26]	16 [11–23]	17 [11–26]	0.090
Activity level [% of day]	7 [0–50]	7 [0–28]	9 [0–35]	0.056

programmed maximal tracking rate was similar between the three groups (*P* = NS).

Anti-PMT algorithm performance

118 patients had at least one PMT episode with a median of 40 episodes per patient. Out of all 46.375 PMT episodes, we analyzed 784 EGMs. An overview of the anti-PMT algorithm performance is shown in Table 2. In the RYTHMIQ group, the diagnosis of PMT was correct in the majority of episodes (124/154) which resulted in a specificity of 80%. The usually reported triggers for PMT such as premature atrial contractions (PACs) and premature ventricular contractions (PVCs) were together responsible for less than one-third of PMT episodes (PAC 11% and PVC 20%). An example of PMT triggered by PAC is shown in Fig. 1 (EGM and schematic) while Fig. 2 shows PMT induced by two consecutive PVCs. In the majority (69%) of correctly diagnosed PMT episodes, the onset of PMT was directly related to the prolongation of the AV delay associated with the AV Search+ protocol (as part of the RYTHMIQ™ algorithm). Fig. 3 shows an example of PMT in the context of AV hysteresis during AV Search+. This algorithm may be on its own responsible for the PMT induction but the risk is increased when premature atrial or ventricular contractions occur during AV hysteresis.

The algorithm incorrectly labeled PMT in 20% of cases in response to normal tracking of intrinsic atrial events at the maximal tracking rate during sinus tachycardia. This occurs in patients where RYTHMIQ previously executed a mode switch from AAI to DDD mode so that atrial sensed events trigger ventricular pacing, a required component for PMT. Fig. 4 shows an example of a patient in sinus tachycardia at maximal tracking rate where PMT was incorrectly diagnosed by the device and which results in a non-conducted P-wave due to the extension of the PVARP after the 16th cycle. In the other patient groups (AV block group and CRT group), the incorrect diagnosis of PMT during sinus tachycardia at maximal tracking rate was responsible for the majority of episodes (66% and 74%, respectively). The correctly diagnosed PMT episodes (specificity of 44% in AV-block patients and 26% in CRT patients) occurred in the context of premature atrial or ventricular contractions. In dependent patients (AV block group) incorrect PMT labeling resulted into an inappropriate PVARP extension and therefore in non-conducted P-waves while in non-dependent CRT

Table 2
Diagnostic overview of the PMT algorithm for the three patient groups. When available, up to 10 PMT episodes were analyzed per patient. Categorical variables are expressed as absolute numbers (percentages). PVC: premature ventricular contraction. PAC premature atrial contraction.

	RYTHMIQ	AV-block	CRT	*P*-value
	n = 156	*n* = 76	*n* = 95	
Patients with PMT episodes	27 (17%)	30 (39%)	61 (64%)	<0.001
Total amount of PMT episodes	13.379	18.699	14.297	
PMT episodes analyzed	154	214	416	
Incorrect PMT diagnosis	30 (20%)	142 (66%)	306 (74%)	<0.001
Correct PMT diagnosis	124 (80%)	72 (44%)	110 (26%)	
PAC [*n*, % of correct]	14 (11%)	54 (82%)	32 (29%)	<0.001
PVC [*n*, % of correct]	25 (20%)	12 (18%)	64 (58%)	<0.001
RYTHMIQ [*n*, % of correct]	85 (69%)	–	–	

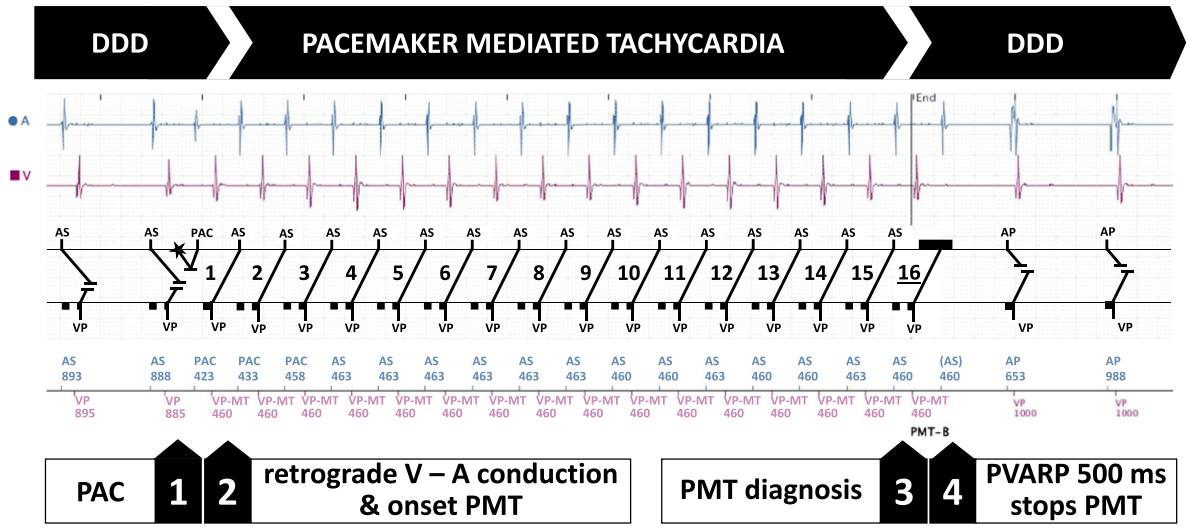

Fig. 1. Example of pacemaker-mediated tachycardia in the context of premature atrial contraction. [1] A premature atrial contraction (PAC) triggers a ventricular pace at the maximal tracking rate (VP-MT). [2] The triggered VP results in retrograde V-A conduction which is sensed as a PAC and on its turn, again triggers a VP (PMT). [3] PMT is diagnosed after 16 stable VP-AS at maximal tracking rate. [4] After the 16th VP-MT, the PVARP is extended to 500 ms which terminates the PMT.

patients the inappropriate therapy resulted in intrinsic ventricular conduction and therefore loss of resynchronization therapy. We have also observed a certain number of phenomena which were less frequent. In the CRT group, inappropriate intervention of the algorithm during exercise was associated with sustained interruption of biventricular pacing in 6 patients. In these patients having sinus tachycardia and with prolonged PR intervals, the intervention (extension of PVARP) resulted in an intrinsic ventricular beat where due to the left bundle branch block the RV sense was also delayed so the succeeding sinoatrial event occurred during the following PVARP. Fig. 5 shows an example where this sequence of sinoatrial events occurring in the PVARP of the preceding intrinsic ventricular events caused extended loss of CRT. In one patient, the inappropriate intervention of the algorithm may have been pro-arrhythmogenic. Fig. 6 shows that directly after the PVARP extension a ventricular tachycardia (VT) is initiated. This patient was equipped with an ICD and the VT was treated with anti-tachycardia pacing. While three burst treatments were ineffective, a ramp treatment succeeded into VT termination.

Discussion

PMT remains a common phenomenon among patients with cardiac stimulation devices. This study permits us to draw important conclusions about PMT in general and about the specificities of the Boston Scientific algorithm.

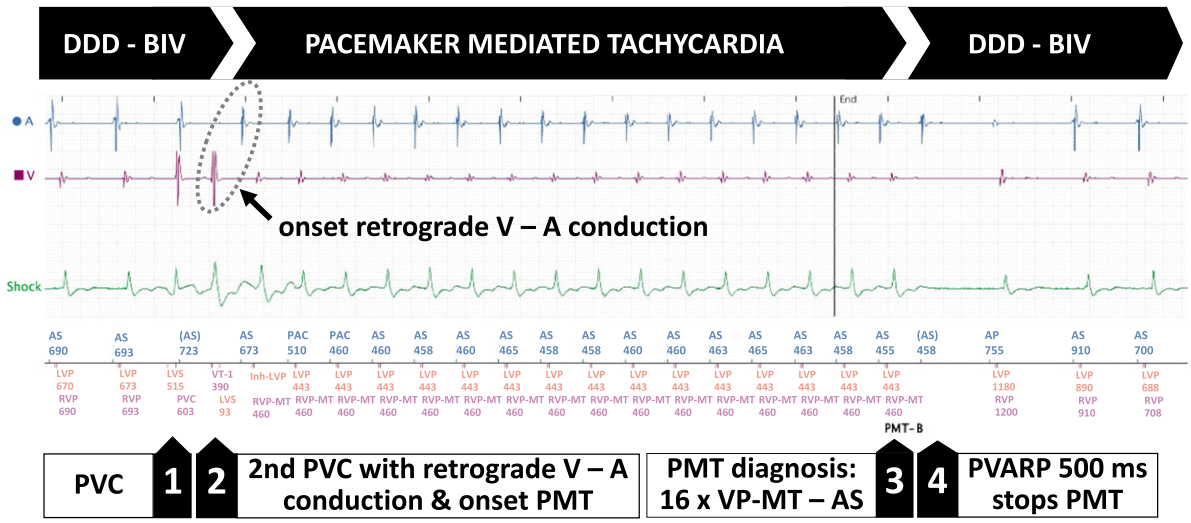

Fig. 2. Example of pacemaker-mediated tachycardia in the context of premature ventricular contraction (PVC). [1] The first PVC occurs simultaneously with a sinoatrial event, the refractory AV node inhibits retrograde (V-A) conduction. [2] However, the second PVC initiates retrograde conduction which results in AS and an AV delay for BIV stimulation. The RV is stimulated at the maximal tracking rate while LV stimulation is inhibited (Inh-LVP) since it is too soon after the preceding LVS. [3] After PMT diagnosis, [4] a single prolongation of the PVARP to 500 ms terminates the PMT.

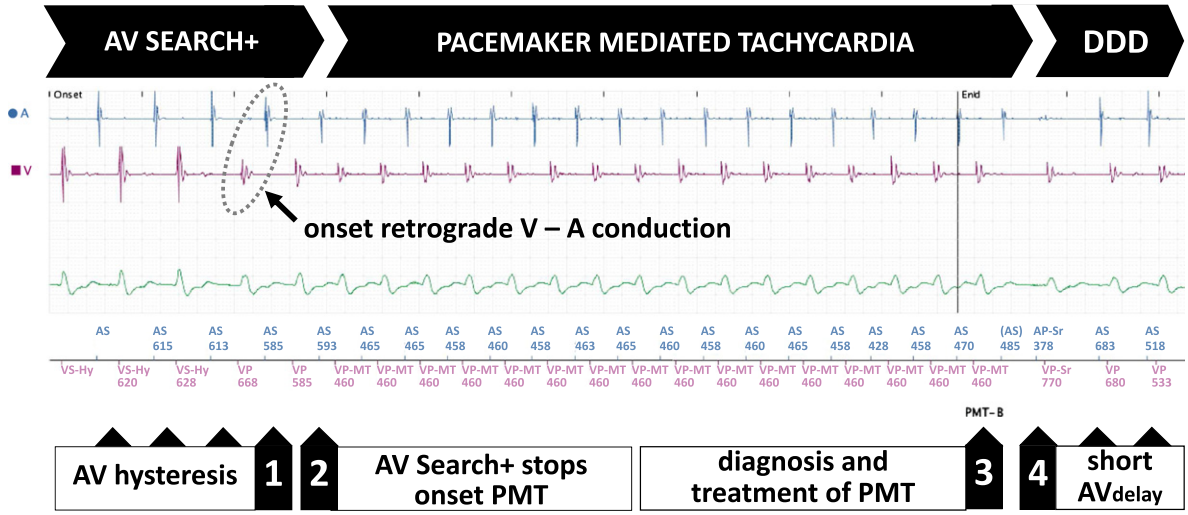

Fig. 3. Example of pacemaker-mediated tachycardia in the context of AV Search+™ (RYTHMIQ™). [1] During AV Search+, the AV delay is extended to search for intrinsic ventricular conduction (marked as VS-Hy). [2] After the third VS-Hy, intrinsic conduction is too slow which triggers a VP. After 2 VPs in a sliding window of 10 the AV Search+ protocol is terminated and the device returns to DDD mode. However, because there is AV block and a prolonged AV delay, the AV-node has recovered by the time the ventricular stimulus occurs enabling retrograde V-A conduction and the ventricle is stimulated at maximal tracking rate (VP-MT). [3] The electronic re-entry is sustained until the pacemaker correctly labels the event as PMT (16 cycles of VP-MT) and increases the post-ventricular atrial refractory period (PVARP) to 500 ms for one cycle which terminates the PMT. [4] Since the AV Search+ phase was terminated, ventricular pacing continues at short AV delay.

Overall performance of the anti-PMT algorithm

The heterogeneous performance of the Boston Scientific anti-PMT algorithm between patient groups indicates that its accuracy is highly dependent on underlying patient etiology. The overall specificity of the anti-PMT algorithm is low (39%) which is caused by incorrect diagnosis of PMT during sinus tachycardia at the maximal tracking rate, especially in patients with a disabled RYTHMIQ algorithm (AV-block group and CRT group). Even though sinus tachycardia at exactly the maximal tracking rate (with variance of at most a few milliseconds) seems to be an unlikely event and the overall incidence of sinus tachycardia is unknown, the sheer number of episodes we found with this issue (478) actually reveals it is quite a common trigger. The other 306 episodes were 'true' PMTs (correctly diagnosed) which were associated with premature atrial contractions (especially in the AV block group), premature ventricular complexes (especially in the CRT group) and surprisingly by the RYTHMIQ algorithm (exclusively in the RYTHMIQ group).

AV Search+

The AV Search+ protocol is part of the RYTHMIQ algorithm which aims to lower the ventricular pacing burden by favoring intrinsic conduction. During AV hysteresis (or

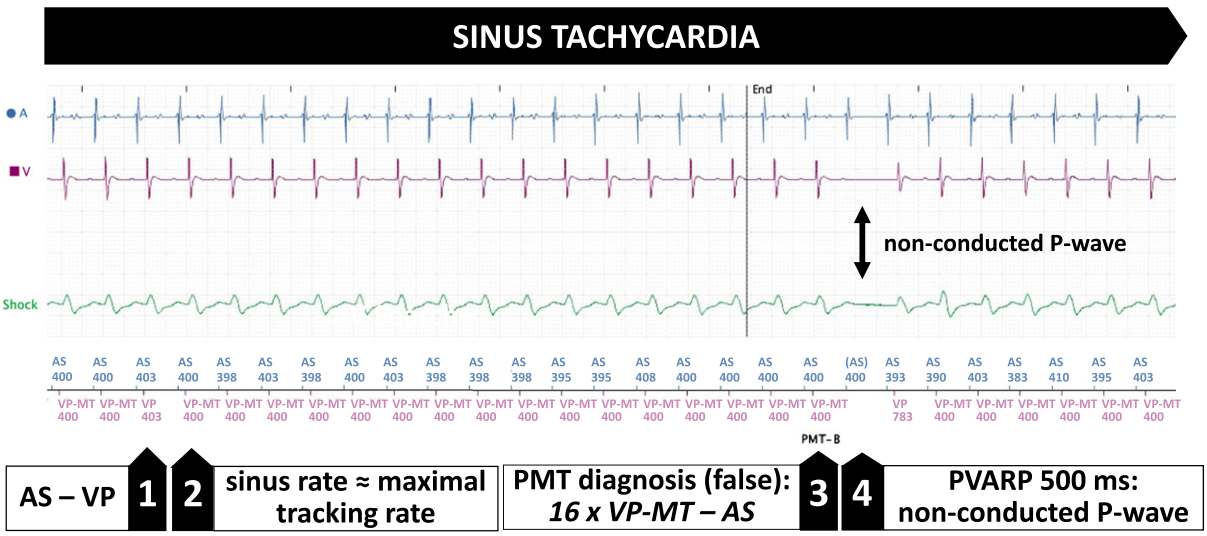

Fig. 4. Incorrectly labeled 'PMT' episode in the context of sinus tachycardia at maximal tracking rate. [1] Atrial sensing during sinus tachycardia close to the maximal tracking rate (≈150 bpm). [1] When the atrial coupling interval is just above maximal tracking (403 ms > 400 ms), a regular VP resets the PMT counter (0/16). [3] Sixteen atrial events at (or slightly faster than) maximal tracking trigger sixteen VP-MT-AS which establishes the diagnosis of 'PMT'. [4] Like all PMTs, the 16th VP-MT is followed by a PVARP extension to 500 ms during which a true atrial sense occurs which is not followed by a ventricular stimulation (non-conducted P-wave).

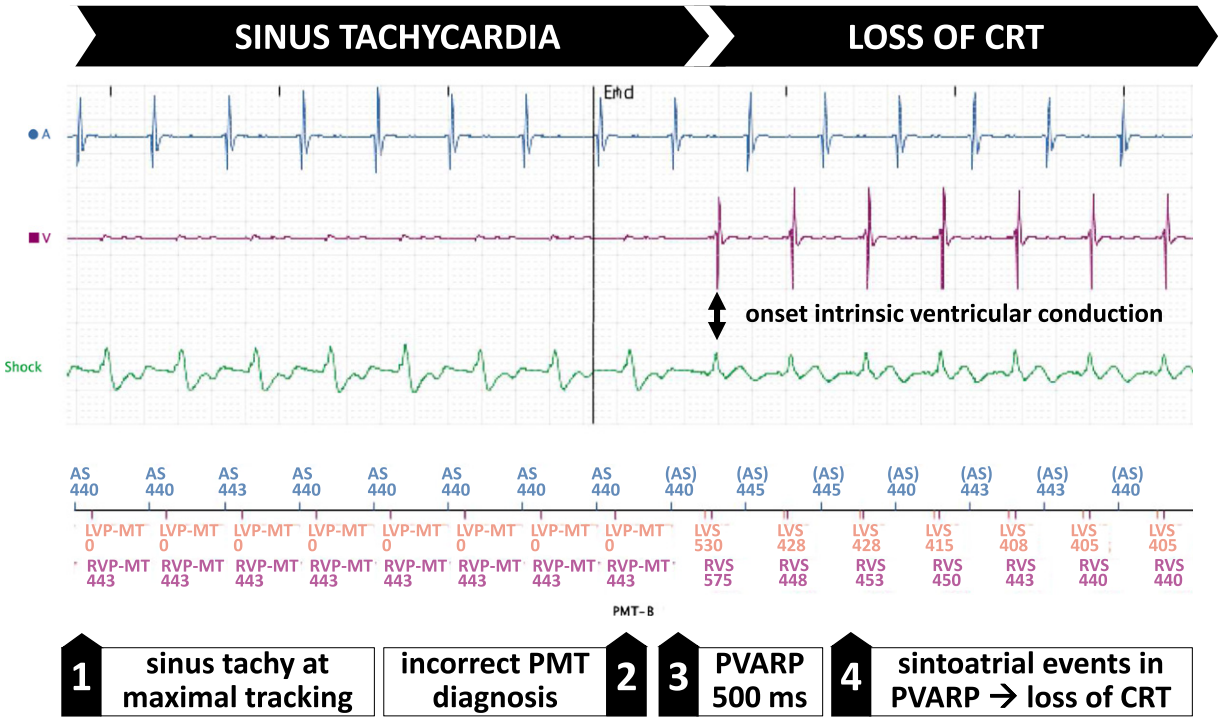

Fig. 5. Example of loss of biventricular stimulation in the context of inappropriate PMT therapy. [1] Patient is in sinus tachycardia which approximates maximal tracking rate. [2] After sixteen sequences of VP-MT-AS the device inappropriately declares PMT. [3] The PMT algorithm results in extension of the PVARP to 500 ms during a single cycle which induces intrinsic ventricular conduction with a relatively long PR interval. [4] Since atrial coupling cycle is short and PR interval is long, every atrial event occurs during the PVARP which induced extended period of loss of biventricular stimulation.

one-step extension of the AV delay) the device determines if intrinsic ventricular events follow atrial events. In some patients, intermittent AV block and a prolonged AV delay permit recovery of the AV refractory period which enables retrograde VA conduction. The possibility of retrograde VA conduction due to a long AV delay may be present at only certain moments of time. Previously, a case report has described the occurrence of PMT associated with this type of algorithm (St. Jude Medical) [11].

We report here the initiation of pacemaker-mediated tachycardia (PMT) by the normal function of a RYTHMIQ AV Search+ algorithm in a large number of patients. This study demonstrates that the link between a prolonged AV delay and initiation of PMT is not rare and was previously underreported. The extension of the AV delay on its own may initiate a PMT (such as shown in Fig. 3) but a combination with a PAC or PVC, which derails atrial and ventricular activation, is also a possible trigger of PMT. The high rate of patients with enabled RYTHMIQ™ and PMT exposes the weakness of using this type of algorithm. Other algorithms do not search for spontaneous conduction by extending the AV delay but rather perform a direct mode switch to AAI (Sorin and Medtronic). This decreases the risk of PMTs while the risk of non-conducted P waves is increased which can be symptomatic in some patients. During DDD mode the AV Search+™ algorithm searches for spontaneous conduction every 32 beats. Progressively increasing this amount may considerably reduce the amount of PMTs. Of important note, after disabling the RYTHMIQ algorithm, the AV Search+™ algorithm remains active and needs to be manually disabled. When not switched off, AV Search+™ will continue to search every 32 beats, even when in permanent DDD mode.

Loss of resynchronization by the ant-PMT algorithm

For a good response to CRT, sustained and adequate biventricular stimulation is imperative [12,13]. The function of a CRT device during a heart rate above the maximal tracking rate depends on the quality of the AV-conduction of the patient. In a resynchronized patient with complete AV-block and preserved chronotropic capacity, a Wenckebach phenomenon occurs when the sinoatrial rate accelerates above the maximal tracking rate. When the sinoatrial rate accelerates above the maximal tracking rate in a resynchronized patient with preserved AV conduction and good chronotropic capacity, the intrinsic activation wave front will dominate ventricular activation and ventricular stimulation is interrupted. In both cases, biventricular stimulation will be re-initiated when the heart rate falls below the maximal tracking rate. However, our study does reveal certain cases of prolonged loss of biventricular stimulation in patients with prolonged intrinsic PR-intervals. The intervention during sinus tachycardia by the PMT algorithm causes a non-conducted P-wave (since the atrial sense occurs within the PVARP) followed by an intrinsic ventricular beat. During exercise, the RP interval is short which increases the risk of the following atrial event to again occur during the PVARP, even though the PVARP is shortened automatically. This phenomenon can result in sustained loss of biventricular stimulation even when the sinoatrial rate falls below the maximal tracking rate. As long

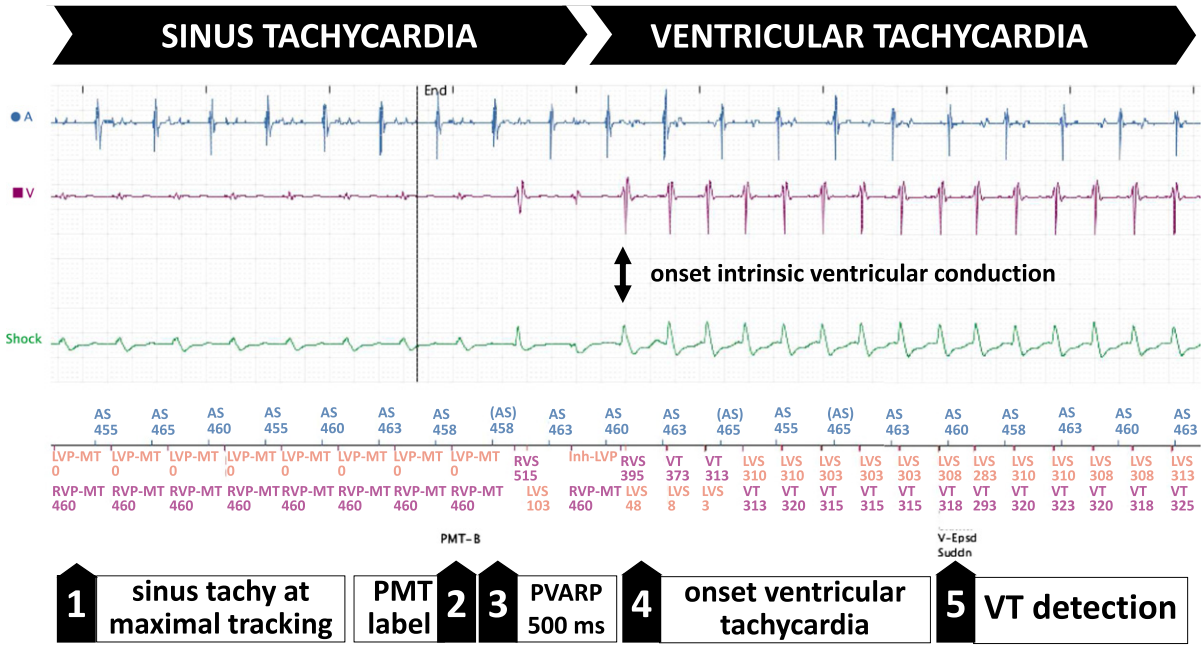

Fig. 6. Example of ventricular tachycardia in the context of inappropriate PMT treatment. [1] Sinus tachycardia at maximal tracking rate, [2] after 16 beats of RVP-MT-AS the device classifies PMT and [3] for a single cycle, the PVARP is extended to 500 ms. [4] After an intrinsic ventricular event (ventricular stimulation is inhibited since the atrial sense occurs during the PVARP) and a stimulated right ventricular event (inhibition of left ventricular pacing), a monomorphic ventricular tachycardia arises. [5] After 8 intervals in the VT zone, the ICD detects the tachycardia and labels "V-Epsd" with criteria of sudden onset ("Suddn").

as the V-A interval occurs during the PVARP, biventricular stimulation will be withheld. Resynchronized patients with long PR-interval need careful programming of the maximal tracking rate and consideration of disabling the anti-PMT algorithm.

Possible arrhythmogenesis

The "short-long-short" ventricular sequence is a well-known phenomenon responsible for the initiation of polymorphic ventricular arrhythmias in long QT syndromes and monomorphic VTs in ICD patients. A case report in the context of a similar device model has previously shown the development of a VT after an inappropriate intervention by the anti-PMT algorithm during sinus tachycardia [14]. The combination of exercise, release of catecholamines and the short-long-short sequence is the probable responsible factors for the induction of the arrhythmias described in the report and in our study. This is the first study that provides data to assess the frequency of the problem. Since inappropriate intervention during sinus tachycardia is common, and we only observed one patient with an associated VT, one may consider it a rare problem. VT induction in pacemaker patient may be more dangerous than in a defibrillator patient, even though their arrhythmogenic risk would probably be lower. To avoid recurrence, it is essential to raise the maximal tracking rate which decreases the risk of inappropriate intervention by the algorithm during sinus tachycardia.

Non-detection of PMT under the maximal tracking rate

The heart rate during PMT depends on a combination of the retrograde conduction time, the programmed maximum rate and the AV delay. An important note is that PMTs below the maximum tracking rate will not meet Boston Scientific's algorithm criteria for PMT, will therefore not be saved to the memory as an episode and will not be treated. This causes difficulties in programming the maximal tracking rate since patients with relatively prolonged retrograde V-A conduction risk developing relatively slow PMTs which will not be diagnosed by the device when the maximal tracking rate is programmed too high. The optimal setting of this parameter must, therefore, strike a balance between the needs of the patient with respect to 1:1 tracking to prevent a Wenckebach periodicity during effort (need to program a high maximal tracking rate) on the one hand, and the risk of non-detection and termination of a PMT (need to program a low maximal tracking rate) on the other hand.

Specificities of the Boston Scientific algorithm

This study is only made possible because a sequence of events classified as PMT is saved into the memory of the device. This is also the case for St. Jude Medical devices but not for Medtronic, Biotronik and Sorin devices. A definite comparison of the PMT algorithms between pacemaker manufacturers is not possible and the results cannot be extended to other algorithms but there are some observations to be made. For the suspicion phase, 8 consecutive AS-VP cycles are required for the devices of all the manufacturers, except for Boston Scientific, which requires 16 cycles, causing a slight delay in the diagnosis and a prolongation of the episodes. During suspicion only the criteria of stability between V-A intervals needs to be met, there is no confirmation phase such as modification of the AV delay

to differentiate between sinus tachycardia, atrial tachycardia and PMT. This explains the low specificity in stimulated patients (dependent or resynchronized patients) with the risk of at least one non-conducted P wave in dependent patients or loss of resynchronization in CRT patients. The algorithms of Biotronik, Saint Jude Medical and Sorin devices are included with a confirmation phase with modulation of the AV delay and analysis of the following VP-AS interval, which limits the risk of intervention for sinus tachycardia during exercise. Saint-Jude Medical also relies on AV hysteresis to search for intrinsic conduction which is a potential trigger for PMT.

Limitations

This study has several limitations, namely the retrospective nature of the analysis and the limited proportion of PMT episodes with stored EGMs (in relation to the total number of PMT episodes). Nevertheless, based on the large amount of episodes we can conclude that the specificity of the algorithm is low. Calculating the sensitivity was not possible since we were not able to investigate PMTs which did not trigger the algorithm to diagnose PMT and save the episode to the device.

Conclusion

Algorithms to minimize ventricular pacing can occasionally have unintended consequences such as PMT. In Boston Scientific devices, the absence of confirmation phase leads to a high rate of incorrect diagnosis of PMT during exercise resulting in non-conducted P waves and loss of biventricular stimulation. The pro-arrhythmogenic risk (long–short cycle) seems limited.

Author contributions

- Strik M: data analysis/interpretation, drafting article, critical revision of article
- Frontera A: critical revision of article
- Eschalier R: data collection
- Defaye P: data collection
- Mondoly P: data collection
- Ritter P: data collection
- Haissaguerre M: data collection
- Ploux S: critical revision of article, statistics
- Bordachar P: concept/design, drafting article, critical revision of article

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